Polychlorinated Biphenyls: Their Effects on Penned Pheasants*

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Introduction

Polychlorinated biphenyls (PCBs) have been useful industrial products since the beginning of their commercial production in 1929. The first report of PCBs occurrence in wildlife was made from the work of Sören Jensen, a Swedish Chemist, in the *New Scientist* in 1966 (1). Since then PCBs have been reported to be widespread in the world's ecosystem, building up in food chains as has been reported for organochlorine insecticides. The chemical structure of PCBs and their action on organisms, although less toxic, are similar to that of DDT. The contamination of human milk, poultry feed, and all classes of wild vertebrates, has created a concern for the need to determine possible harmful effects of PCBs.

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Because of the economic importance of the pheasant (*Phasianus colchicus*), it was chosen as the experimental animal using PCBs in this study. The objectives were (1) to determine the patterns of absorption, storage, and excretion for PCBs, and (2) the effects of PCBs on reproduction, behavior, and survival. Since many published reports indicated that the chromatograms of PCBs in wildlife most nearly resemble those of Aroclor 1254, this was the PCB product chosen for study.

The use of brand names in this paper does not indicate an endorsement of any product.

Materials and Methods

Aroclor 1254 (supplied by the Monsanto Co.) was mixed (w/w) in a 1:9 dilution with corn oil and weighed into No. 00 size gelatin capsules to within ±2.5 percent of the required weight. Capsules were administered into the esophagus using a glass tube. Capsules containing only corn oil were administered to control birds.

Analyses for residues were made by Dr. Yvonne A. Greichus, Experiment Station Biochemistry Department, South Dakota State University, and the Bureau of Sport Fisheries and Wildlife laboratory at the Denver Wildlife Research Center. Analytical procedures used at the South Dakota State University laboratory are described by Dahlgren et al. (2) and procedures of the Denver Laboratory by Dahlgren et al. (3).

Hens for breeding experiments were purchased in the winters of 1970 and 1971 (30 and 34 respectively) from the South Dakota Pheasant Co. of Canton, and cocks were raised from birds previously obtained from the Canton source. All birds used for breeding were about 1 year of age.

In late January they were placed under 16 hours daily of artificial light to stimulate breeding. Both sexes were fed a pheasant breeder ration (Zip Feed Mills, Sioux Falls). One tablespoonful of oyster shells was added to the food cup of each hen weekly. Cocks were kept in breeder cages (30×36×18 inches high), and hens were kept in individual cages (12×18×12 inches high) designed to facilitate handling and identification of eggs. Temperatures were kept at near 65 F in colder months. Eggs were collected daily, individually numbered, set weekly for 15 weeks in a forced-draft incubator, and hatched in pedigree cages.

In 1970, cocks were first dosed with 25 mg PCBs on February 13, and weekly thereafter for a total of 17 times. Hens were first dosed 1 week later, February 20. Breeding was commenced February 16. Eggs were gathered for the first weekly egg group from March 7–13 and gathered last 15 weeks later, June 13–19. In 1971, the same sequence was followed, except that the first dosing began February 19 for cocks and February 25 for hens, and breeding began March 1. Eggs were gathered for 15 weekly egg groups beginning March 6 and ending June 18.

Within 36 hours after hatching, chicks were taken from the incubator, wingbanded, and one wing was removed to the alula. They were then placed on a visual cliff (4) and given 5 minutes to jump to either the visually deep or shallow side. Chicks from this study and those hatched from a concurrent dieldrin study were placed together in brooders and fed a turkey pre-starter diet (Zip Feed Mills) until they were 6 weeks of age. At 6 weeks, they were placed outside in 16×16 foot pens and fed a pheasant grower ration (Zip Feed Mills).

Several times in summer and fall, chicks were caught by hand by the same person, and wingband numbers were recorded. The number of birds caught in each category in the first and last half of all birds caught was compared with an expected 50:50 distribution using chi-square analysis. Each pen was a closed unit, and birds of several groups caught predominantly in the first half caused birds of other categories to be caught in the last half. Weights to the nearest gram were taken of all adult breeders weekly. Chicks were weighed to the nearest gram at 6 weeks of age.

To study effects of PCBs in combination with dieldrin, cocks and hens from 6 to 9 months old were stratified by sex and weight and randomized to five groups of 22 birds each. They were given capsules containing either (1) 4 mg technical grade dieldrin, (2) 8 mg dieldrin, (3) 50 mg PCBs, (4) 100 mg PCBs, or (5) 50 mg PCBs and 4 mg dieldrin together. Ten doses were given, one capsule each 3.5 days for 5 weeks. Three and one-half days after the last capsule was given, all surviving birds were sacrificed.

The Denver Wildlife Research Center analyzed for residues of PCBs in pooled livers from: three road-killed pheasants collected near Washington, Pennsylvania; six road-killed pheasants collected southeast of Lancaster, Pennsylvania; six pheasants shot east of Gary, Indiana; six pheasants shot in Benton County, Indiana; and six pheasants shot near Brookings, South Dakota. All of the above collections were made in the spring of 1971.

Results and Discussion

Effects on Reproduction

The averages of eggs laid per hen per day among hen treatment groups were significantly different in 1970 (5). With the use of an orthogonal test, the rate of egg production among hens given 12.5 mg PCBs was not found to be lower ($P>0.05$) than the rate of the control group; but the rate of the 50-mg group was lower ($P<0.01$) than the rates of the other groups. In 1971, the same pattern was evident (Table 1); there were significant differences ($P<0.05$) among hen groups. The production of hens given 12.5 mg was not lower ($P>0.05$) than that of controls, while production of 50-mg hens was lower ($P<0.01$). Egg production was lower in groups where cocks were given PCBs, but this is not to be expected, and we cannot explain it.

Differences among groups in egg fertility in 1970 were significant ($P<0.05$); however, variation in fertility did not follow a pattern related to PCB dosage, and, thus, the results may not have any biological meaning (5). In 1971, there were no significant differences in fertility among groups (Table 1).

Hatchability in both 1970 (5) and 1971 was highest in control groups and lowest in groups in

Table 1. Reproductive statistics from control pheasants and pheasants given PCBs 1970–71.

Treatment group[a]	No. eggs per hen per day	No. eggs set in incubator	Fertile eggs		Fertile eggs hatched		Fertile eggs pipped but not hatched	
			No.	Percent	No.	Percent	No.	Percent
1970								
0–0[b]	0.621	277	101	36	74	73	6	6
0–12.5	0.459	220	128	58	87	68	29	23
0–50	0.292	136	50	37	32	64	8	16
25–0	0.335	168	91	54	73	80	9	10
25–12.5	0.362	179	71	40	50	70	18	25
25–50	0.198	98	63	64	41	65	10	16
1971								
0–0	0.337	168	82	49	65	79	6	7
0–12.5	0.436	199	128	64	64	50	20	16
0–50	0.328	148	96	65	51	53	4	4
25–0	0.465	233	170	73	116	68	8	5
25–12.5	0.385	288	153	53	109	71	15	10
25–50	0.228	95	42	44	10	24	5	12

[a] Each treatment group had five hens, except that the 25–12.5 mg group had nine hens in 1971.
[b] The first number is the weekly PCB level in mg given to cocks; the second, that for hens.

which hens had received 50 mg PCBs. In 1970, these differences tested by chi-square were not significant ($P>0.05$), while in 1971 hatchability was reduced ($P<0.01$) among hens given PCBs. Significant differences ($P<0.05$) were found in the number of eggs that were pipped but not hatched for hen groups in both 1970 and 1971. Apparently, the administration of PCBs to the hen adversely affected the viability of the embryo at hatching. McLaughlin et al. (6) injected both 10 and 25 mg of Aroclor 1242 into chicken eggs and found only 0–5 percent hatchability with growth retardation, edema, and beak deformities in embryos.

Eggshell thickness was measured only in 1970, and no significant differences ($P>0.05$) in eggshell thickness were found using analysis of variance (5). Eggshells from hatched eggs (without membranes) laid by control hens averaged 0.26 ± 0.02 mm standard deviation, and those from all hen groups receiving PCBs averaged 0.23 ± 0.02 mm. Eggshells from unhatched eggs averaged 0.32 ± 0.02 mm for control hens, and 0.31 ± 0.02 mm for hens receiving PCBs. Dahlgren and Linder (7) found the eggshell thickness of pheasants to be unaffected by weekly administration of capsules containing 6 mg dieldrin.

Heath et al. (8) fed 25 ppm of Aroclor 1254 to mallards (*Anas platyrhynchos*) through two breeding seasons. They also fed bobwhite quail (*Colinus virginianus*) 50 ppm PCBs and at a joint level of 25 ppm PCBs and 15 ppm DDE for one reproductive season. They found no effects on egg production, cracked eggs, eggshell thickness, embryonation, embryos alive at 3 weeks, normality of hatchlings, and normal hatchlings alive at 14 days.

Scott et al. (9), who fed 0, 0.5, 1.0, 10.0, and 20.0 ppm Aroclor 1248 for 8 weeks to chickens in full egg production, found no reduction in egg production on the lowest levels of PCBs after 8 weeks; however, they noted a 10 percent reduction was associated with 10 ppm, and a 13 percent reduction was associated with 20 ppm. They reported that 10 ppm Aroclor 1248 reduced hatchability of chicken eggs by 8 percent after 4 weeks and 44 percent after 8 weeks. The 20 ppm level nearly eliminated all hatchability. Most embryos died at 21 days of development; many after pipping. They also found that eggshell strength was not affected when as much as 20 ppm was fed.

Effects on Behavior

Seventy-one 11-week old hens given one 210-mg

Table 2. Behavior on the visual cliff of chicks hatcheP from control pheasants and pheasants given PCB's 1970–71.

Treatment group	No. that jumped to visually deep side		No. that jumped to visually shallow side		No. not jumping within 5 minutes	
	1970	1971	1970	1971	1970	1971
0–0[a]	2	9	35	43	8	7
0–12.5	3	7	42	34	8	5
0–50	3	7	6	30	5	6
25–0	5	16	33	67	15	17
25–12.5	5	16	22	78	7	7
25–50	4	0	5	9	6	1

[a] The first number is the weekly PCB level in mg given to cocks; the second, that for hens.

capsule in the evening of the first day of testing appeared weak at the end of the following day. After receiving the second capsule, they sat with feathers fluffed, and some tremoring was noticed. During the 24 hours before death, they often showed tremors, particularly when disturbed. Shortly preceding death, birds were comatose and died without tremors.

Eleven 11-week old hens given 20 mg daily were hyperexcitable after 4 days of PCB treatment; after 10 days they sat with feathers fluffed. They exhibited weakness with occasional tremors about 30 days after dosage began. Eleven hens 11 weeks old given 10 or 20 mg daily appeared to have fewer body feathers after 30 days than controls, although they did not appear to peck one another more often. Flick et al. (10) reported feather loss in chicks of the domestic chicken given PCBs.

Visual Cliff Behavior—In 1970, when chicks were placed on a visual cliff for up to 5 minutes, significant differences ($P<0.01$) among groups were found in their behavior using chi-square analysis (5); offspring of hens given 50 mg jumped to the visually-deep side of the cliff more often than offspring in other groups. In 1971, no significant differences in behavior between groups were noted (Table 2). When data from both years were combined, no significant differences ($P>0.05$) were found. Baxter et al. (11) reported that pheasant chick behavior in a visual-cliff test was apparently affected by dieldrin given their parents.

Response to Hand Catching—Results of catching young pheasants by hand were analyzed by comparing the number of birds from each treatment group caught in the first half and last half of all birds caught to an expected number equaling 50 percent of the birds in that group. For example, 16 out of 20 young in the category where both parents had received PCBs were caught in the first half of all birds caught on July 22, 1970 (Table 3). The 16 caught in the first half and 4 caught in the second half were compared by chi-square analysis to an expected 10 (half the total category of 20), resulting in a highly significant difference ($P<0.01$). Since each pen was a closed unit and the PCB-treatment birds were penned with other young from a concurrent dieldrin study, it must be remembered that birds of several groups caught predominantly in the first half would cause birds of other groups to be caught in the last half. It is important to compare not only how the PCB birds were caught in comparison to an expected distribution, but also how categories compared with one another. The comparisons are shown in Table 3 as ratios. These indicated that the ability of penned pheasants to avoid hand capture was significantly less in 1970 offspring where both parents had been given PCBs. This is identical to the findings of Dahlgren et al. (12) for offspring of parents administered dieldrin. However, in 1971, the responses of offspring of treatment groups were similar to controls.

Data for 1970 and 1971 were combined and a contingency table chi-square technique was used to determine the effect of PCB treatment on the response to hand catching between groups. The response was similar whether PCBs were given to either the cock or hen but was significantly different ($P<0.05$) when both parents received PCBs from that where only one parent received PCBs.

During catching, pheasants were herded around and around the pen, and often the catcher was able to capture those birds which ran back directly toward the pursuer. However, there was no attempt made to determine or to quantify the elements of behavior that would explain why offspring of birds treated with PCBs were easier to catch.

Table 3. Effects of PCBs on hand capture of penned pheasants, 1970–71. (Numbers represent birds caught in the first half of all birds caught; numbers in parentheses represent one-half the number of that category in the pen. Chi-square was used to compare numbers actually caught with half of the numbers in each category.)

Dates of capture	No. of hatches caught	No. of pens	Parents receiving PCB			
			Both	Hens only	Cocks only	Neither
1970						
July 22	1–8	14	16(10)**	8(8.5)	7(10.5)	11[a](10.5)
July 29	1–9	15	19(11.5)**	7(10)	9(12)	6(13.5)**
Aug. 13	1–10	18	11(12.5)	12(12.5)	12(13.5)	8(13)*
Aug. 19	1–12	20	21(15.5)*	17(14)	18(17.5)	19(18)
Sept. 1	1–14	22	25(18)*	19(15.5)	22(20)	14(19.5)
Oct. 15	1–15	23	20(16)	14(16)	25(17.5)*	9(14)
Nov. 21	1–15	13	14(11.5)	14(12.5)	8(10.5)	12(11.5)
Dec. 5	1–15	13	11(11)	14(11.5)	12(10.5)	6(10)**
All 1970 catches combined			137(106)**	105(100.5)	113(112)	85(110)**
Ratio, 1970[b]			1.55	1.91	1.98	2.59
1971						
July 21	1–7	17	27(19.5)*	23(24.5)	25(21.5)	12(14.5)
Sept. 1–3	1–15	28	75(70.5)	41(37)	39(36.5)	21(22)
Sept. 21–23	1–15	28	71(70.5)	38(36)	37(36)	24(21.5)
Oct. 7–11	1–15	28	33(37)	33(35.5)	27(35)	19(20.5)
Oct. 19–22	1–15	28	34(35.5)	23(33.5)*	35(34.5)	17(21)
Nov. 2–5	1–15	28	30(30.5)	29(32.5)	23(28.5)	19(18)
All 1971 catches combined			270(264.5)	187(199)	187(192)	112(117.5)
Ratio, 1971			1.96	2.13	2.05	2.10
All 1970–71 catches			407(370.5)**	292(299.5)	300(304)	197(227.5)**
Ratio, 1970–71			1.82	2.05	2.03	2.31

[a] Totals of row numbers may exceed one-half total birds because odd numbers of birds in pens were rounded higher.
[b] Birds in pen/birds caught in first half.
* ($P<0.05$).
** ($P<0.01$).

Table 4. Weights and survival for the first 6 weeks of offspring from control pheasants and pheasants given PCBs, 1970–71.

Treatment group	Average weight at 6 weeks (g)		No. of chicks to brooder		No. of chicks alive after 6 weeks		Percent survival		
	1970	1971	1970	1971	1970	1971	1970	1971	Both years
0–0[a]	396	429	73	65	49	50	67	77	72
0–12.5	378	435	84	64	49	45	58	70	64
0–50	303	425	29	50	3	37	10	74	51
25–0	389	428	72	115	50	91	69	79	75
25–12.5	403	458	50	109	30	72	60	66	64
25–50	344	373	40	10	9	6	22	60	30

[a] The first number is the weekly PCB level in mg given to cocks; the second, that for hens.

Table 5. Survival to the fall of offspring of control pheasants and pheasants given PCBs, 1970–71.

Group	No. of young alive in fall[a]		Percent survival from 6 weeks of age to fall[b]			Percent survival hatching to fall[c]		
	1970	1971	1970	1971	Both Years	1970	1971	Both Years
0–0[d]	20	36	41	72	57	27	55	41
0–12.5	23	36	47	80	63	27	56	40
0–50	0	29	0	78	72	0	58	37
25–0	21	57	42	63	55	29	50	42
25–12.5	18	59	60	82	75	36	54	48
25–50	4	4	44	67	53	10	40	16

[a] December 5, 1970; November 2–5, 1971.
[b] Using the number of chicks alive after 6 weeks, Table 4.
[c] Using the number of chicks to brooder, Table 4.
[d] The first number is the weekly PCB level in mg given to cocks; the second, that for hens.

Effects on Survival and Weights of Offspring

Survival—Chick survival was determined for treatment groups in 1970 (5) and 1971 during the first 6 weeks of age while chicks were kept in brooders (Table 4). The survival of chicks was not related to whether cock parents received PCBs, but chi-square analysis showed that significantly more ($P<0.01$) deaths occurred among chicks hatched from hens receiving 50 mg PCBs in 1970. These differences were not evident in 1971 data. When data from both years were combined, there was a significant difference ($P<0.01$) between offspring where hens received 0 and 12.5 mg PCBs and between offspring where hens received 12.5 mg and 50 mg PCBs ($P<0.01$).

Survival of young pheasants in outdoor pens was measured in December, 1970, and November, 1971 (Table 5). Survival of offspring from 6 weeks of age to the fall in both years appeared to be unaffected by level of treatment in either year. When overall survival from hatching to the fall in both years was considered, the overall survival of offspring from hens given 50 mg was significantly less ($P<0.05$) than that of the other groups. The lower rates of survival for the 50-mg groups were due to the effect on early survival, not that from mortality of birds older than 6 weeks of age. No meaningful departures from the expected 50:50 sex ratios in the treatment groups were determined (Table 6). Apparently PCBs did not affect the survival of one sex more than another.

Weight—Weights of chicks from hens on 50 mg PCBs weekly were lower ($P<0.01$) at 6 weeks of age (5) than those of other groups in 1970 (Table 4). In 1971, weights of offspring of 50-mg hens were lower ($P<0.01$) than that of controls, while weights of offspring of 12.5-mg hens were higher ($P<0.01$). When data from both years were combined, there was no relationship between weight and treatment level.

Although we did not adequately determine that

Table 6. Numbers of cocks and hens alive in November that were offspring of control pheasants and pheasants given PCB's, 1970–71. (Chi-square was used to compare numbers of each sex alive with an expected 50:50 distribution.)

Group	1970 November 21		1971 November 2–5		1970–71 Combined	
	Cocks	Hens	Cocks	Hens	Cocks	Hens
0–0[a]	11	11	17	17	28	28
0–12.5	14	11	15	20	29	31
0–50	0	1	13	17	13	18
25–0	5	16*	33	22	38	38
25–12.5	13	6	36	20*	49	26**
25–50	1	1	3	1	4	2
All PCB groups	33	35	100	80	122	115

[a] The first number is the weekly level in mg of PCBs given to cocks; the second, that for hens.
* ($P<0.05$).
** ($P<0.01$).

PCBs via the egg depressed the weight of offspring, McLaughlin et al. (6) mentioned growth retardation of embryos as an effect of PCBs injected into the yolk sac of chicken eggs. We fed no pheasant chicks PCBs, but Flick et al. (10) found that 1-day-old chicks fed Aroclor 1242 at 200 and 400 ppm had depressed growth by the second week of feeding and that the growth depression was related to the level fed. Vos and Koeman (13) fed PCBs to 1-day-old cockerels and found body-weight depression from 400 ppm Aroclor 1260. Platonow and Funnell (14) found that 1-day-old cockerels fed 250 ppm Aroclor 1254 had depressed body weights between the sixth and ninth week of their feeding trial; this depression was associated with reduced feed consumption. Rehfeld (15) also found depressed weight gains in 1-day-old chicks fed sublethal levels (10–50 ppm) of Aroclor 1254; chicks fed 30 and 50 ppm for 2.5 weeks and then fed a clean diet recovered from the growth depression, while chicks fed 40 and 50 ppm for 5 weeks and then fed a clean diet for 8 weeks did not show a recovery from the growth depression.

Body weights and Mortality in Birds Given PCBs

Adult hens in both the 1970 and 1971 breeding experiments were unaffected in body weight by administration of as much as 50 mg PCBs in single capsules weekly. It was characteristic, however, of birds that died on PCB treatment to stop eating and die within several days.

Hens 11-weeks old given a 210-mg capsule of PCBs in the evening ate very little the following day and appeared weak by the end of that day (3). After receiving the second capsule 24 hours subsequent to the first, birds sat with feathers fluffed; some tremoring was noticed; and no food was consumed. Birds of both sexes 6–9 months of age given 100 mg or 50 mg PCBs every 3.5 days for 5 weeks continued to eat, as did 11-week-old hens repeatedly given either 10 or 20 mg daily. Scott et al. (9) found no effect on feed consumption when laying chickens were fed up to 20 ppm Aroclor 1248, and no mortality that could be attributed to treatment. Prestt et al. (16) found no effect on weight of Bengalese finches (*Lonchura striata*) when they were fed up to 400 ppm Aroclor 1254 for 56 days.

Birds 11-weeks old given daily capsules containing 210 mg PCBs died within 1.3 and 5.9 days; the 16 designated to be analyzed upon death lived longer than the other birds in the experiment, from 2.2–5.9 days, averaging 3.8 ± 1.0 day standard deviation (3). They lost from 15–37 percent of their initial weight before death. All control birds lived. Correlations of the initial weight, days to death, and percentage weight loss were obtained for 53 birds in this experiment. Initial weight was correlated with days to death ($r = 0.699$, $P < 0.01$); heaviest birds lived longest. The birds which were heaviest initially lost the greatest percentage of their weight before death ($r = 0.589$, $P < 0.01$). Birds that lived the longest lost the greatest percentage of their weight before death ($r = 0.744$, $P < 0.01$). Time of death of the 11 birds that were not given PCBs, but were starved, ranged from 2.3 days to 8.6 days, averaging 3.9 ± 1.8 days. They lost from 27–51 percent of their weight by the time of death (3).

Of the birds given 50 or 100 mg every 3.5 days for 5 weeks, 4 of 22 died in the 50-mg group and 7 of 22 died in the 100-mg group. In addition, two birds in the 100-mg group were so weak they were near death at the conclusion of the experiment.

Mortality of birds given 10 mg began 30.6 days after the first capsule was given; the ninth bird died after 179.3 days. The other seven birds died between 50.3 and 60.6 days after initial treatment. The tenth bird of this group, still alive after 8 months of treatment, was sacrificed (3).

In the 20-mg group, the first bird died 39.6 days and the last bird 54.1 days after capsules were first given. The average number of days to death was 46.1 ± 5.3 days (3).

Mortality was light in breeding experiments in 1970 and 1971. In 1970, only 2 hens died from among the 30 hens and 10 cocks under study; both hens had received 50 mg PCBs weekly (5). In 1971, among 34 hens and 14 cocks in the study, three hens died, two that had received 12.5 mg and one that had received 50 mg weekly.

Tucker and Crabtree (17) reported that 2000 mg/kg of either Aroclor 1242, 1254, 1260, or 1268 given to mallards caused no mortality or symptoms. Prestt et al. (16) estimated that 254 mg/kg/day given to Bengalese finches would give 50 percent mortality at 56 days. Heath et al. (8) reported that Aroclor 1254 had an LC_{50} of 1090

ppm when fed for 5 days as part of the diet to pheasants. Vos and Koeman (13) reported that, of 20 chicks fed 400 ppm for 60 days, only three died.

In the present study, 210 mg PCBs daily were given to 11-week-old hens. By the time the average bird died, it had received 840 mg PCBs, the first to die received 420 mg, and the last to die, 1260 mg. These same figures for 11-week-old hens on 20 mg daily were 940 mg, 800–1100 mg; and for birds given 10 mg daily (one was sacrificed after surviving 8 months) were >830 mg, 310–2410+ mg. Thus, for a group of pheasants given PCBs at 10 mg or more daily, the most susceptible individuals would probably die with 300–400 mg Aroclor 1254; the average bird would die with a cumulative dose totaling from 800 to 950 mg; and the least vulnerable would die after receiving 1200–2410+ mg PCBs.

Absorption, Storage, and Excretion

Dahlgren et al. (2) reported that retention of PCBs in the bodies of four hens, each given single 50-mg capsules and sacrificed 28 days later, averaged 40.5 mg in each hen. Levels of PCBs were highest in all tissues at 12 hours after capsule administration and declined most rapidly in liver at 24 hours. Throughout their experiment, levels were highest in liver, followed by brain and muscle. Brain tissue contained more PCBs than muscle per unit weight, but, having a higher lipid percentage than muscle tissue, brain tissue had a smaller concentration of PCBs per unit of lipid.

Analysis of whole bodies of four hens that received 12.5 mg PCBs weekly for 17 weeks revealed that they retained from 37 to 56 percent of the administered dose, while hens on the 50-mg level retained 60 to 82 percent of the administered dose. The hens on the higher dose averaged about six times as much PCBs in their bodies as the hens on the lower dose, although they had been given only four times as much PCBs (2).

A total of 48.7 mg of PCBs in the whole body plus that excreted in eggs and feces over 28 days was accounted for after administration of a 50-mg capsule (2). The PCB-corn oil mixture was readily absorbed in the gut of the pheasant; calculations showed that a maximum amount excreted unabsorbed was 6 percent of the 50 mg given, and as much as 98 percent may have been absorbed.

Five of the hens that had received 12.5 mg weekly for 16 weeks in the 1971 breeding experiment were sacrificed 1 week following the administration of the last capsule in the series. The ppm PCBs in the bodies of these five hens were 17.6, 18.6, 24.4, 28.3, and 30.5, averaging 23.8 ppm. Three months later, three other hens of the same group, which had been kept caged, were sacrificed. The analyses of their bodies showed 8.9, 12.0, and 20.0 ppm PCBs, averaging 13.6 ppm PCBs. After 6 months on a clean diet, three other birds similarly treated had whole-body ppm values of 18.3, 19.5 and 25.0, averaging 20.9 ppm. The apparent rise in PCB concentration at 6 months may be due to sample variation and/or the physiological state of the hens sampled. It is obvious that the rate of excretion of PCBs is relatively slow. These birds were analyzed at the Denver Wildlife Research Center.

Scott et al. (9) found that chicken hens given up to 20 ppm Aroclor 1248 in the diet for 8 weeks lost less than 50 percent of the stored PCBs after 4 weeks on either a standard diet or low-energy diet. He also found that a low-energy diet followed by a high-energy diet did not affect reduction of PCBs over time. Prestt et al. (16) fed 1500 mg Aroclor over 56 days to Bengalese finches and were able to recover only 9 percent of the amount fed, when they analyzed one bird from the experiment.

Excretion Via the Feces—Excretion of PCBs in feces was relatively low as reported by Dahlgren et al. (2). An average of 4.0 mg per single 50 mg dose was excreted in the feces of four hens over 28 days; the feces from these birds were analyzed as a pool. Two other hens excreted 2.2 and 2.9 mg per 50-mg dose over a 28-day period; the feces from these two hens were analyzed separately. PCBs in the pooled feces of the four hens were at a peak during the first week and declined to relatively lower levels thereafter. They also found that excretion in the feces was highest during the first 24 hours in the two other hens given single 50-mg capsules, and less than 1 mg PCBs appeared in the feces the first day. Variability in excretion between these two hens was low. An average of 2.6 mg per hen was passed in the feces of the two hens by the end of 28 days.

Excretion Via the Egg—In four hens given 50-mg capsules when egg laying was declining, levels of PCBs in the eggs were lowest in the first

week, highest during the second week, and declined thereafter. The average excretion per hen via the egg was calculated to be 4.2 mg PCBs (2). Excretion of PCBs via the egg could be higher than excretion through the feces when hens are in full egg production. A single egg laid between 1 and 2 weeks after a hen was administered a single capsule containing 50 mg PCBs was shown to contain 1.5 mg of PCBs. This egg was one of two laid that week by one hen.

Heath et al. (8) reported that two mallard eggs from the second reproductive season, when birds had been on 25 ppm Aroclor 1254, had 56 ppm and 33 ppm PCBs, wet weight. Peakall (18) reported that ring dove (*Streptopelia risoria*) eggs taken from birds given 10 ppm Aroclor 1254 in the diet averaged 4.81±1.08 ppm standard error. The actual amounts in mg of PCBs in ring dove (18), mallard (8), and pheasant eggs in the present study would be far below the 10 mg injected by McLaughlin et al. (6) into chicken eggs. The 10 mg resulted in poor hatchability, edema, and beak deformation.

Scott et al. (9) found that PCBs deposited in chicken eggs were less than 0.5 ppm after 8 weeks with 0.5 and 1.0 ppm Aroclor 1248 in the diet. They found levels of over 3 ppm after 8 weeks with 10 ppm in the diet and levels of about 6–7 ppm in eggs of hens on 20 ppm. These values are much lower than those found in the present study from 3–4 weeks after administration of a single capsule of 50 mg Aroclor 1254. Their results, showing a drastic reduction in hatchability associated with 7 ppm or less residue in eggs, are not comparable to our findings with pheasants. Differences with the experimental animal or the Aroclor product used may have resulted in the gross differences in findings between our studies. Heath et al. (8) reported a nearly four-fold difference between bobwhite and Japanese quail in the LC_{50}; thus species differences may be important.

Residues in eggs of wild birds have been determined by several authors from diverse collection points. Anderson et al. (19) found PCBs in all egg pools of cormorants (*Phalacrocorax auritus*) and pelicans (*Pelecanus erythrorhynchos*); cormorants were shown to have an estimated 8 ppm in eggs and pelicans 0.6 ppm. Jensen et al. (20) reported 48 ppm in the egg of a heron (*Ardea cinerea*) collected in Sweden, and 8–21 ppm from 9 guillemot (*Uria aalge*) eggs from the Baltic Sea. Risebrough et al. (21) reported that an egg of a peregrine falcon (*Falco peregrinus*) contained 10 ppm; 8 black petrel (*Loomelania melania*) eggs had an average of 1 ppm; 5 eggs of a barn owl (*Tyto alba*) had <1 ppm; and a golden eagle egg (*Aquila chrysaetos*) had <1 ppm. Dustman et al. (22) reported median measurements of egg residues of bald eagles to be 1.65 ppm for Alaska and 9.7 for those from all other states. They further reported median egg levels of 15.9 ppm for the osprey (*Pandion haliaetus*) of Connecticut and 2.5 ppm for those in Maryland; 5–6 ppm in brown pelican (*P. occidentalis*) eggs from different areas; and 5 ppm in eggs of royal terns (*Thalasseus maximus*).

Prestt et al. (16) found residues ranging from 0–80 ppm in 363 eggs from 28 species of both land and water birds collected in Britain. Most of these had less than 10 ppm, but those which had higher levels included a single egg of the great crested grebe (*Podiceps cristatus*), 40 ppm; one egg among 101 of the heron that had 80 ppm, while the arithmetic mean was 5 ppm; one egg of a moorhen (*Gallinula chloropus*) with 15 ppm, among 13 samples that averaged 2.4 ppm; and a single egg of a great skua (*Sterocorarius skua*) that had 25 ppm.

Particularly in view of findings that about 5 ppm was associated with nearly complete negation of hatchability in the chicken (9), the above findings in some wild birds are alarming. Apparently, though, as earlier pointed out, there must be considerable differences among species in (1) the rate at which they deposit PCBs in the egg, and (2) what a particular ppm range may mean in associated deleterious effects.

Residue Levels in Tissues

We analyzed tissues from seven different groups of birds: (1) 16 birds given 210 mg PCBs daily that were designated for analysis upon death, (2) five additional birds on 210 mg daily that died, (3) nine birds that were killed at intervals for matching with the 16 designated to die, (4) pooled samples of birds killed 12 hours and 24 hours after receiving a single capsule containing 210 mg PCBs, (5) pooled samples of four birds dying and from four birds surviving capsules con-

taining 50 and 100 mg PCBs, (6) a pooled sample of four birds dying on 20 mg and 10 mg PCBs, and (7) a pooled sample of two control birds (3).

Birds that died from daily doses of 210 mg PCBs had brain levels that ranged from 320 to 770 ppm wet weight and averaged 520 ppm±110 ppm standard deviation. Liver residue levels were much more variable than brain levels. They ranged from 390 to 9,300 ppm and averaged 2,500±2,000 ppm wet weight. Muscle residues were also relatively more variable than brain residues, as they ranged from 51 to 290 ppm, and averaged 140±53 ppm. Tissue levels from the birds killed for matching overlapped with ranges in the birds that died on 210 mg, but less so for brain than for liver or muscle ranges. Brain residue levels of nine birds sacrificed for matching ranged from 280 to 500 ppm, and averaged 370±65 ppm; livers ranged from 1,000 to 5,000 ppm, and averaged 1,900±1,300 ppm; and muscle ranged from 58 to 110 ppm, and averaged 83±17 ppm.

Brain showed less variability than other tissues. Further, brain was the only tissue independent of other parameters (initial weight, percent weight loss, days to death, and lipid content in brain, liver, and muscle) when testing correlations of all possible parameters using birds that died from capsules containing 210 mg PCBs. This lack of correlation with other parameters indicated the usefulness of the brain in assessing toxic levels of PCBs. The relationship of the liver with other parameters such as original bird weight and days to death tended to negate its usefulness. Further, the interrelationships of liver parameters with those of muscle, even though it is not known how many of these relationships were meaningful, tended to negate the usefulness of muscle as an indicator tissue.

Brain levels of PCBs were generally higher in birds that died than in birds that were sacrificed at the same time for matching of brain residue levels, although there was some overlap of the two groups. A brain residue level of 400 ppm separated the bulk of the birds (86 percent) that died from most (67 percent) of those that were sacrificed. There appeared to be a relationship between days to death and brain residue level; however, this was not significant ($P>0.05$). A pooled sample of four birds that died on treatment with 100 mg PCBs given each 3.5 days had 320 ppm in the brain tissue, while a pooled sample of four sacrificed birds in this group had 59 ppm. A pooled sample of three birds that died on treatment with 50 mg PCBs given every 3.5 days had 350 ppm in the brain, while a pooled sample of four sacrificed from this group had 34 ppm. Pooled brain tissues from four birds that died on daily doses of 10 and 20 mg had 360 and 380 ppm PCBs, respectively; no data were available for survivors of these groups. These latter data indicated that 300 ppm might be better than 400 ppm as a separation point that would indicate death from PCBs and that, when smaller amounts of PCBs were received by the birds over a period of time, the spread in brain levels between birds that died and survived might be greater.

Ratios of residue levels using wet-weight ppm values among brain, liver, and muscle in dead and sacrificed birds overlapped considerably, particularly in brain:liver and brain:muscle ratios. However, liver:muscle ratios appeared to be smaller in birds dying on 210-mg doses. If one establishes 19 as a liver:muscle ratio, 3 of 9 birds sacrificed on the 210-mg doses had smaller ratios and only 4 of 18 birds dying had higher ratios. However, it is necessary to gather more data to determine if this ratio is useful in diagnosing the cause of death as PCB toxicosis.

Vos and Koeman (13) found that, when 1-day-old cockerels were fed 400 ppm Phenochlor DP 6, Clophen A60, and Aroclor 1260 in the diet for 60 days, the chicks had brain residue levels ranging from 70 to 700 ppm among 11 birds that died on treatment and 40 ppm for one chick which survived. Liver levels in their experiment were more variable than brain levels and ranged from 120 to 2,900 ppm among 28 birds that died and from 210 to 340 ppm among four survivors. Seven of their 28 birds that died had liver residues of less than 250 ppm, while four of 11 birds had less than 300 ppm. It appears from their data that liver might be as useful as brain for diagnosing cause of death. Their liver:brain ratios were similar to those of the present study, but, since they had only one survivor from which brain and liver were analyzed, overlap could not be evaluated. Rehfeld (15) reported that 30–50 ppm Aroclor 1254 given to 1-day-old cockerels resulted in liver residues of 300–500 ppm. Prestt et al. (16) reported a range

of 3–634 ppm in livers of survivors and 70–697 ppm in livers of Bengalese finches dying during treatment with Aroclor 1254, and that liver level was correlated with the amount received ($P<0.01$).

Prestt et al. (16) stated that the liver:brain residues should be compared and that brain ppm/liver ppm X 100/1 was three times higher in Bengalese finches that died during their experiment than in birds killed at the end. Data in the present study showed a complete overlap in brain ppm/liver ppm X 100/1; birds that died during testing had a range in ratios of 5.4 to 112.8, while matching birds sacrificed had 10.0 to 37.0.

Relatively little sampling of brain tissue for PCB residues has been done in wild birds. Dustman et al. (22) reported that a sick bald eagle had 230 ppm PCBs in its brain, and that PCBs may have contributed to its death. Jensen et al. (20) reported that three white-tailed eagles (*Haliaeetus albicilla*) had a brain residue range of 29–70 ppm PCBs, averaging 47 ppm; muscle residues ranged from 150–240 ppm, averaging 190 ppm. Risebrough et al. (21) found 0.04, 1.5, 21, and 34.6 ppm PCB in the brains of four peregrine falcons. Prestt et al. (16) reported liver residues ranging from 0 to around 900 ppm from a wide variety of British birds; arithmetic averages ranged from 0.5 ppm in the buzzard (*Buteo buteo*) to 98 ppm in the heron. The tissue levels reported above were below those at which mortality occurred in the present study, except that the muscle level for the white-tailed eagle was higher than those in this study associated with death in the pheasant from PCBs. However, these levels from wild birds do constitute substantial percentages of the brain and liver levels in the present study where death occurred from PCBs.

Histopathologic Effects

Dahlgren et al. (3) found that PCBs decreased weights of heart and spleen at all treatment levels ($P<0.01$). PCB treatment increased weights of kidney and liver in birds given 10- and 20-mg doses ($P<0.01$), but no effect was seen in the 210-mg group. Starved birds had smaller hearts ($P<0.05$) and livers ($P<0.01$) than controls.

Splenic atrophy, as described by Flick et al. (10) and Vos and Koeman (13) was found in all pheasants given 20 mg daily and in the 10-mg birds except for one that survived 8 months and was sacrificed (3). Splenic atrophy was characterized by almost complete absence of lymphatic nodules and an increase in the relative abundance of red pulp. In one of the birds given 20 mg, loci of necrosis were found in lymphatic nodules.

Prestt et al. (16), using the Bengalese finch, reported that kidneys were larger in birds that died from PCBs than in controls. Flick et al. (10), using chickens, mentioned both enlarged adrenals and kidneys from PCB treatment. In our study, kidneys were larger in pheasants given 10 or 20 mg daily, but neither kidney nor adrenal enlargement was visually detected during necropsy. McCune et al. (23), using Aroclor 1242 with chickens, mentioned both enlarged livers and kidneys in birds given PCBs. Platonow and Funnell (14) and Rehfeld (15), using 1-day-old chicks, reported enlarged livers with dietary intake of Aroclor 1254. Grant et al. (24) reported enlarged livers and decreased spleen size over a period of time in the rat. Flick et al. (10) and Vos and Koeman (13) reported small spleens in their studies. Although hydropericardium was found in varying degrees by many of the authors cited, it was found only rarely in the present study. Vos and Koeman (13) found that Phenoclor DP 6 and Clophen A60 caused much more liver necrosis and hydropericardium than Aroclor 1260; this was probably due to contaminants (25).

PCBs in Combination with Dieldrin

Mortality among pheasants of both sexes 6–9 months old given dieldrin, PCBs, or a combination of the two, varied with the level of chemical administered (Table 7). Among the 22 pheasants on each level, 3 died with 4 mg dieldrin per capsule, and 6 died with 8 mg dieldrin. The same proportions held true for PCBs, since 4 died with 50 mg PCBs per capsule, and 9 died with 100 mg PCBs. When 50 mg PCBs and 4 mg dieldrin were administered together, a total of 9 birds died. None of 11 control birds died during this period of time. These data suggest that effects of PCBs and dieldrin together are additive, not synergistic. Heath et al. (8) found that the joint toxicity of Aroclor 1254 and DDE given to Japanese quail was additive, and found no evidence of synergism in their joint effect.

Table 7. Mortality occurring among 22 pheasants of both sexes that were 6–9 months of age when PCBs and dieldrin were administered separately and in combination. No mortality occurred among 11 control birds.

Group treatment	No. of deaths in week: 1	2	3	4	5	Total deaths
50 mg PCBs	1	0	0	1	2	4
100 mg PCBs	0	3	0	4	2[a]	9
4 mg Dieldrin	3	0	0	0	0	3
8 mg Dieldrin	3	0	0	1	2	6
50 mg PCBs and 4 mg Dieldrin	1	2	2	0	4	9

[a] These two birds were near death at the end of the experiment.

Residues in Wild Birds

A pooled sample of six pheasant livers taken from wild South Dakota pheasants had <0.1 ppm PCBs. A pooled sample of three livers collected near Washington, Pennsylvania, had <0.1 ppm PCBs, and a pooled sample of six livers collected southeast of Lancaster, Pennsylvania, had 2.0 ppm PCBs. A pooled sample of six livers collected east of Gary, Indiana, had 0.5 ppm PCBs, and a pooled sample of six livers taken from pheasants in Benton County, Indiana, had 1.5 ppm PCBs.

These relatively low levels in livers of wild pheasants taken both from industrial areas and rural areas indicate these pheasants were probably exposed to small amounts of PCBs. Prestt et al. (16) found that residue levels in birds in Britain were related to their food habits. They found the most PCBs in livers of fresh-water fish-eating birds (up to about 900 ppm); bird-feeding raptors had up to 70 ppm; birds which eat mammals had up to 50 ppm; birds with a mixed diet of mammals, birds, and carrion had up to 15 ppm; and those which eat insects had 0–1 ppm.

Summary and Conclusions

PCBs given to laying pheasant hens adversely affected egg production, hatchability, and viability of the embryo about the time of hatching but did not affect fertility or eggshell thickness. The chief effects on reproduction occurred through the PCBs taken in by the hen, not by the cock.

A subtle effect on behavior was indicated by studies on the visual cliff and of the ability of offspring to avoid hand capture. These behavioral effects have been reported for birds with organochlorine insecticides and could be deleterious to a wild species, in that instinctive patterns necessary for survival are involved. Behavioral differences may be affected through PCB administration to the cock as well as through the hen, as shown by the study of hand catching; this implies that the effect is, though unexplained as to mechanism, more than through the physical presence of the PCBs in the egg-yolk lipids. The presence of PCBs in egg lipids is probably responsible for the observed effect of increased mortality in young during the first 6 weeks of life, and in a possible depression of weight at 6 weeks of age. Only early survival of chicks was affected, as no effect was noted in survival from 6 weeks of age through the fall. Sex ratios of young pheasants surviving to the fall showed an expected 50:50 distribution, thus PCBs did not affect the survival of one sex more than another.

Large doses of PCBs, 210 mg daily, effected a loss of appetite, but lesser doses tested did not affect feed consumption until just prior to death. The death of birds given PCBs was not attributable to the starvation that occurred in the few days prior to death, because birds dying from PCBs did not lose as much weight as birds which were not given PCBs but were starved. Further, starved birds did not show the histopathologic effects observed in PCB-treated birds such as degeneration of liver cord cells and depletion of lymphatic nodules in the spleen.

PCBs were (1) rapidly and readily absorbed into the pheasant's body, (2) stored in the lipid fraction, and (3) excreted slowly in feces and eggs. Excretion via the egg may be an important means of ridding the body of PCBs for the hen, but the PCBs in the egg yolk lipid may be dangerous for the offspring because of altered behavior and lowered survival both of the embryo and hatched young.

Brain tissue may be a valuable indicator of PCB toxicosis; levels of 300–400 ppm or more were associated with death due to PCBs. Marked splenic atrophy was the most consistent characteristic noted among several organ parameters checked in birds that died from PCBs. Enlarged

kidneys and livers were also useful characters in attempting to diagnose PCB toxicosis.

PCBs and dieldrin were not, when combined, synergistic in their joint toxicity to pheasants.

Livers from wild pheasants collected in Pennsylvania, Indiana, and South Dakota, did not exceed 2 ppm PCBs, indicating relatively low-level contamination.

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